British Heart Journal, 1978, **40**, 1143-1148

Effect of prolonged intensive training on cardiorespiratory response in patients with angina pectoris

E. BEN ARI, J. J. KELLERMANN, C. LAPITOD, Y. DRORY, E. FISMAN, AND M. HAYAT

From the Cardiac Evaluation and Rehabilitation Institute, Chaim Sheba Medical Center, Tel Hashomer, Israel

SUMMARY The cardiorespiratory response to prolonged work was investigated in 15 patients (age 51 ± 5·6) after transmural myocardial infarction; the patients had angina pectoris. Based on an individually determined pain threshold heart rate the following two relative work loads were obtained; 55 per cent and 90 per cent of threshold heart rate. Training was monitored using the 10-channel Siemens radio-telemetry system, and consisted of 30 minutes continuous pedalling, twice per week. Pretraining results showed a substantial increase in heart rate (HR 12 ± 8·2) and systolic blood pressure (SBP 15 mmHg) between the 5th and the 10th minute of work and decrease in O_2 consumption (Vo_2 l/min) and O_2 pulse between the 15th and 30th minute of exercise.

Training resulted in the following changes: Decreased heart rate at rest and during work ($P \leqslant 0{\cdot}01$). Systolic blood pressure did not rise up to the 15th minute of work. Oxygen consumption increased gradually, reaching a steady state after 15 minutes of work. O_2 pulse increased gradually and remained constant during the last 15 minutes of work. SBP × HR product decreased significantly ($P \leqslant 0{\cdot}05$-$0{\cdot}01$) at rest and during work. Favourable changes in minute ventilation and ventilation equivalent indicate improved respiratory adjustment. Clinically there was a pronounced decrease in severity and frequency of angina pectoris along with increased work time before onset of pain. The data show that intensive prolonged training may result in improvement of the physiological adaptive mechanism of patients with angina pectoris to continuous physical stress.

In most of the studies of exercise capacity of patients with angina pectoris, work periods have been short lasting several minutes only (Eckstein, 1957; Jones and Reeves, 1968; Redwood *et al.*, 1972; Kennedy *et al.*, 1976). For many years we have been interested in the physiological effect of prolonged intensive training on the adaptive mechanism to exertion in angina pectoris patients. The aim of the present investigation was to study the cardiorespiratory adjustment to prolonged work periods in angina patients, and to evaluate the effect of the improved physical work capacity on the related measurements.

Subjects and methods

Fifteen men with angina pectoris (aged 51 ± 5·6 yr), 14 after transmural myocardial infarction and one without previous infarction, were under observation for a 4-month period. All patients had typical ischaemic electrocardiographic changes, with 1 mm horizontal ST segment depression during or shortly after physical exertion. These patients had previously participated in a calisthenic-type exercise programme lasting for approximately a year. At the conclusion of this programme, none of the patients showed significant improvement in any of the measured variables related to the cardiorespiratory system. Additional testing was conducted before (T1) and after 4 months (T2) of intensive ergometric training. Since true maximal heart rate and oxygen consumption could not be attained by these patients, a pain threshold heart rate was determined for each patient at the onset of anginal pain (Kellermann, 1975). Two exercise loads were then determined for each patient, the first 55 per cent of each threshold and the second 90 per cent of pain threshold heart

Received for publication 18 May 1977

rate. The following observations were made during exercise testing[1]: heart rate, brachial arterial blood pressure, expired gas collection (Kofranyi respirometer) and electrocardiogram at rest, after 5 minutes of pedalling at 55 per cent of the patient's pain threshold heart rate, after 10 minutes at 90 per cent of the patient's pain threshold heart rate, and after 25 minutes at this latter load. Recovery heart rate and blood pressure were measured 3 and 5 minutes after cessation of exercise. Measurements of O_2 consumption were made at least twice and the results averaged, in order to reduce possible methodological errors.

The following values were calculated:

$$O_2 \text{ pulse} = \frac{O_2 \text{ consumption (ml/min)}}{HR}$$

Ventilation equivalent =

$$\frac{\text{Minute ventilation}}{O_2 \text{ consumption (ml/min)}} \times 100$$

Double product = Systolic BP × HR (Sarnoff *et al.*, 1958)

$$\text{Respiratory quotient} = \frac{CO_2 \text{ produced}}{O_2 \text{ consumption}}$$

Room temperature was maintained at 22 ± 1°C.

The test was discontinued in the event of increasing chest pain, other subjective complaints (dizziness, dyspnoea), additional pathological electrocardiographic adnormalities (ST depression exceeding 3 mm, arrhythmias, multifocal increasing frequency of early cycle ventricular premature contractions), or consistent decrease in systolic blood pressure, or after completion of 30 minutes continuous pedalling.

TRAINING

The *calisthenic programme* included breathing exercises, muscular relaxation, non-competitive ball games, gymnastics, and running twice weekly with an energy expenditure of 2 to 9 cal/min. The annual programme consisted of the following phases:

(1) 1st to 10th week, slow rate low intensity exercises in supine position emphasising continuous breathing and long recovery periods between exercises. Walking at individually convenient rate for 5 to 10 minutes.

(2) 11th to 20th week, introducing some muscle strength exercises, increasing variety of exercises, and increasing speed and duration of walking.

(3) 21st to 30th week, increasing rate and intensity of exercise performed, decreasing recovery time between exercises, short jogging periods (10 to 30 s), and non-competitive games.

(4) 31st to 50th week, increasing time of jogging (1 min) and intensity of floor exercises, exercises in erect position.

[1]*Multistage, near maximal ergometric test.* After detailed physical examination and resting electrocardiogram, the test normally started with a 25-Watt load with subsequent 25-Watt increments. Each phase of work was of 5 minutes duration, followed by a 5-minute rest period. The following age-related target heart rates were used: 20 to 39 years, 170/min; 40 to 59 years, 150/min; 60 years and over, 130/min.

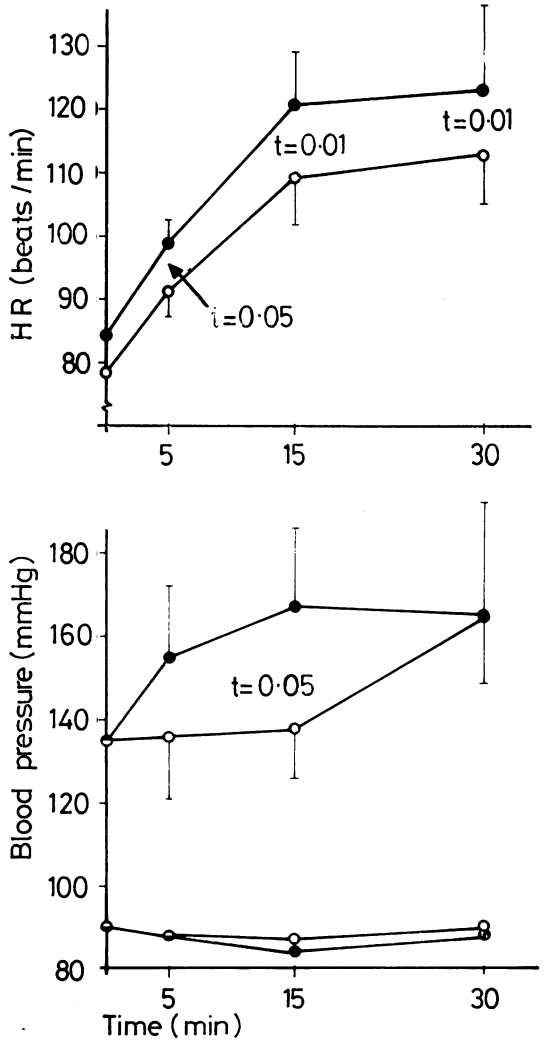

Fig. 1 *Heart rate and blood pressure during prolonged work before* (●———●) *and after* (○———○) *4 months of intensive training.*

Table 1 *Circulatory measurements in patients with angina pectoris before and after a 4-month intensive ergometric training programme*

Load and time	*HR (beats/min)*		*P*	*Blood pressure (mmHg)*		*P*	*SBP × HR*		*P*
	Before	*After*		*Before*	*After*		*Before*	*After*	
Rest	83 ± 11·3	78 ± 8·08	NS	135/90 ± 17·5/9·9	135/90 ± 15·7/8·7	NS	12 057 ± 2 655	10 549 ± 1 296	NS
5 min at 55 per cent of pain threshold heart rate	109 ± 4·0	92 ± 4·0	< 0·01	155/88 ± 17·8/8·0	136/87 ± 15·0/10·0	NS	14 134 ± 1 647	12 191 ± 1 796	< 0·05
10 min at 90 per cent of pain threshold heart rate	121 ± 8·2	109 ± 8·8	< 0·01	167/84 ± 19·4/8·7	138/87 ± 13·7/8·3	< 0·05	20 364 ± 3 018	17 501 ± 2 100	< 0·01
25 min at 90 per cent of pain threshold heart rate	123 ± 14·5	113 ± 8·1	< 0·05	164/87 ± 18·3/8·8	164/89 ± 17·3/8·6	NS	20 916 ± 3 610	18 419 ± 2 333	< 0·01

NS, not significant.

Table 2 *Oxygen consumption (Vo_2) and O_2 pulse in patients with angina pectoris before and after 4-month intensive ergometric training programme*

Load and Time	*O_2 pulse (ml O_2/beat)*		*P*	*Vo_2 l/min*		*P*
	Before	*After*		*Before*	*After*	
Rest	4·2 ± 1·4	4·4 ± 1·04	NS	0·343 ± 0·020	0·342 ± 0·025	NS
5 min at 55 per cent of pain threshold heart rate	7·7 ± 1·8	8·7 ± 1·5	< 0·05	0·764 ± 0·058	0·764 ± 0·065	NS
10 min at 90 per cent of pain threshold heart rate	9·8 ± 1·5	9·9 ± 1·8	NS	1·230 ± 0·101	1·038 ± 0·095	< 0·05
25 min at 90 per cent of pain threshold heart rate	8·9 ± 1·7	9·8 ± 2·0	< 0·05	1·119 ± 0·100	1·100 ± 0·090	NS

NS, not significant.

Ergometric training lasted 4 months and took place twice weekly, always during the morning. Using the Monark bicycle ergometer, the first 5 minutes of pedalling (to 55% of pain threshold heart rate) were considered as 'warm up'. This was followed without a pause by the next load (to 90% of pain threshold heart rate) for 25 minutes. A constant rate of pedal revolutions was not required, but was generally seen to be 50 ± 10 revolutions/min. The patient's electrocardiogram was monitored using the 10-channel radiotelemetry system of Siemens Telecust.

STATISTICAL METHOD

Standard deviation was calculated by methods using the ungrouped data procedure, while the differences between the means and levels of significance were obtained using Student's t test for small samples (Guilford, 1965).

Results

The results are presented in Tables 1 and 2 and Figs 1 and 2.

HEART RATE

The prescribed ergometric training resulted in a significantly ($P < 0·01$) lower heart rate during exercise at similar work loads.

BLOOD PRESSURE

Comparison between T1 and T2 showed that ergometric training did not change systolic blood pressure at rest or at the end of exercise. During exercise in T1, systolic blood pressure rose substantially (20 mmHg) after 5 minutes of work at the warm-up level (55% pain threshold heart rate). Further rises were recorded when the load was increased to 90 per cent and throughout the next 10 minutes. During T2, systolic blood pressure remained essentially constant until the 15th minute of work, after which a significant increase of 25 mmHg was observed (Fig. 1). No changes in diastolic blood pressure were observed during any phase of exercise in either T1 or T2 (Table 1).

O_2 PULSE

Training did not significantly affect oxygen consumption at the individually limited levels (Table 2,

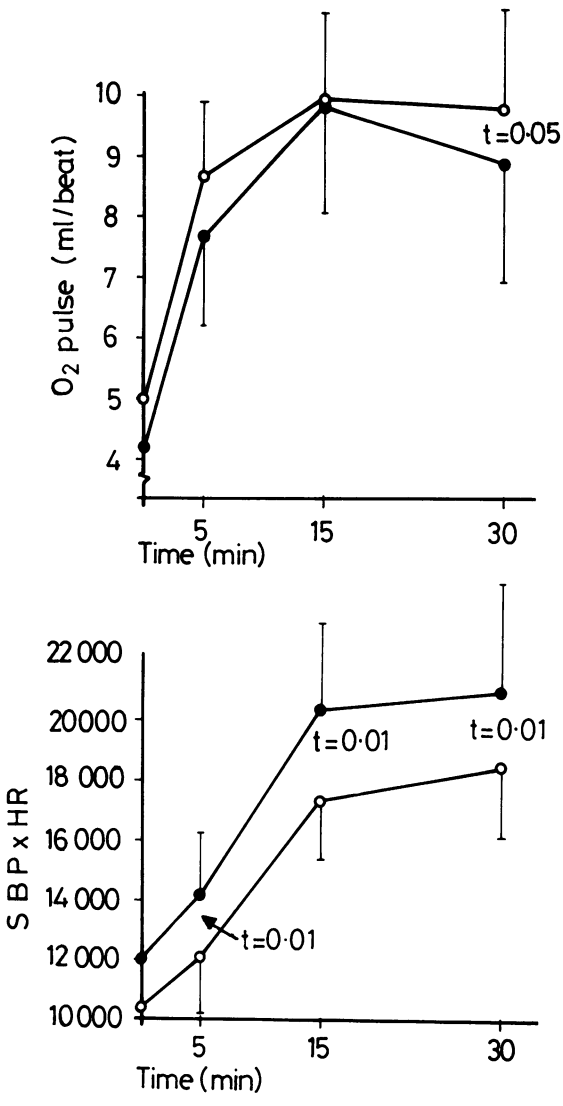

Fig. 2 *Oxygen pulse and double product during prolonged work before (●———●) and after (○———○) 4 months of intensive training.*

Fig. 2). A reduction of 111 ml/min was observed between the 10th and 25th minute of exercise, but only in T1. During T2, oxygen consumption increased gradually, reaching a 'steady state' level after about 20 minutes. Relating oxygen consumption to heart rate, O_2 pulse increased similarly before and after training up to the 15th minute of work (Table 2, Fig. 2). From this point up to the end of exercise, there was a decrease of 0·9 ml in O_2 pulse in T1, while in T2 O_2 pulse remained constant. Systolic blood pressure × heart rate (double product), which is generally accepted as an indirect estimation of the myocardial oxygen consumption (MVo_2) (Sarnoff *et al.*, 1958), was significantly ($P < 0·01$) lower after training (Table 2, Fig. 2). A reduction of about 2000 arbitrary units from T1 to T2, both at rest and during each phase of exercise, reflects a decrease in myocardial work.

VENTILATORY MEASUREMENTS

The ergometric training resulted in slight improvement in ventilation, ventilation equivalent, respiratory quotient, and energy expenditure during the various phases of training, but these changes were not statistically significant.

Discussion

The ability to perform continuous exercise depends basically on adequate supply of oxygenated blood and fuel for combustion for the working muscles. It is well established that cardiac output increases in proportion to work demands and then remains constant, both in healthy sedentary males and in coronary patients (Wade and Bishop, 1962; Ekelund and Holmgren, 1964; Ekelund *et al.*, 1967; Hartley *et al.*, 1969). However, the time course of cardiac output adaptation was found to be faster in healthy subjects (Jones and Reeves, 1968). Though cardiac output values during continuous submaximal work are similar in healthy men and in coronary patients, the total output of oxygenated blood during an exercise period is greater in normal men.

Ekelund *et al.* (1967) reported an increase of 30 beats per minute (bpm) between the 10th and the 60th minute of work. Grimby *et al.* (1966) found an increase of 10 to 12 bpm and Ekblom (1970) 20 bpm during an hour of pedalling. In this study, heart rate increased by 10 to 12 bpm only up to the 15th minute of work. This difference is not significant and can in part be attributed to differences in age, fitness level, and relative work load. As heart rate increases, stroke volume decreases at moderate and even low work loads (Levy *et al.*, 1961; Cobb and Johnson, 1963; Saltin and Stenberg, 1964; Ekelund, 1967; Sowton and Burkart, 1969; Redwood *et al.*, 1972). These circulatory changes, resulting from decreased cardiac filling, which is a consequence of the lower tone of the capacitance vessels (Ekelund and Holmgren, 1964), are basically similar in healthy subjects and in coronary patients.

Data on the favourable effect of training on myocardial function have not been consistent. Several investigators, most of whom have used moderate exercise routines, reported unimproved left ventricular function (Redwood *et al.*, 1972; Kennedy *et al.*, 1976). On the other hand it seems

that prolonged intensive or hypoxic training conditions result in improved myocardial performance (dP/dt max) and triple product (Monroe, 1964; Scheur and Stezoski, 1972; Scheur *et al.*, 1974; Alexander and Liu, 1976; Carey *et al.*, 1976; Sturzenhoffcker *et al.*, 1976). In our study, patients were rarely stopped during exercise. Usually they were told to continue pedalling and were asked to stop only if there were increasing chest pain and/or ST depression exceeding 3 mm. Blood pressure during continuous submaximal exercise has been described in healthy men (Holmgren, 1956; Hartley *et al.*, 1969; Granath *et al.*, 1970). In contrast to healthy subjects, coronary patients in our study showed a steady increase in systolic blood pressure during T1. Training resulted in significantly lower systolic blood pressure from the start up to the 15th minute of exercise, suggesting a reduction in total vascular resistance and contractile work of the left ventricle during this time. It is interesting to note that changes in blood pressure and heart rate were not as pronounced in patients with angina pectoris who were participating in a calisthenics and game-type programme (Kennedy *et al.*, 1976) or other coronary rehabilitation programme reviewed by Detry (1973). True maximal oxygen consumption was not attained by these patients; symptom-limited oxygen consumption increased by 8 to 10 per cent, as in several other studies involving coronary patients (Detry, 1973). The decrease in oxygen consumption during the last 15 minutes in T1 resulted in decreased O_2 pulse. This suggests a decreased blood flow to the myocardium which was not evident after training.

Ventilatory measurements showed slight but not significant changes similar to those obtained after training in healthy men. In addition, it may be assumed that local peripheral changes, increasing the arteriovenous oxygen difference, occurred in our patients also.

Clinically, there was a decrease in the severity and frequency of angina pectoris in almost every patient who underwent ergometric training. The patients described less severe pain with slower progression; they could often continue to work and walk without taking glyceryl trinitrate and could do more work before the onset of anginal pain.

In conclusion, we found that intensive training improved cardiorespiratory function and enabled coronary patients to do prolonged work more efficiently. The improved central and peripheral circulatory adaptation is responsible for the decrease in the physiological response to exercise and consequent relief of anxiety and lack of confidence of untrained angina patients during prolonged exercise. The fact that none of the patients dropped out of the training is attributed to the good physiological response, high motivation, group co-operation, and the excellent relation between the group and the medical team.

The authors thank Mrs P. Nachum, R.N., for her work in supervising the training programme.

References

Alexander, C. S., and Liu, S.-M. (1976). Effect of hypercapnia and hypocapnia on myocardial blood flow and performance in anaesthetized dogs. *Cardiovascular Research*, **10**, 341-348.

Carey, R. A., Tipton, C. M., and Lund, D. R. (1976). Influence of training on myocardial responses of rats subjected to conditions of ischaemia and hypoxia. *Cardiovascular Research*, **10**, 359-367.

Cobb, L. A., and Johnson, W. P. (1963). Hemodynamic relationships of anaerobic metabolism and plasma free fatty acids during prolonged strenuous exercise in trained and untrained subjects. *Journal of Clinical Investigation*, **42**, 800-810.

Detry, J.-M. R. (1973). *Exercise Testing and Training in Coronary Heart Disease*, pp. 9-45. Williams and Wilkins, Baltimore; Stenfort-Kroese, Leiden.

Eckstein, R. W. (1957). Effect of exercise and coronary artery narrowing on coronary collateral circulation. *Circulation Research*, **5**, 230-235.

Ekblom, B. (1970). Effect of physical training on circulation during prolonged severe exercise. *Acta Physiologica Scandinavica*, **78**, 145-158.

Ekelund, L.-G. (1967). Circulatory and respiratory adaptation during prolonged exercise. *Acta Physiologica Scandinavica*, **70**, Suppl. 292, 1-38.

Ekelund, L.-G., and Holmgren, A. (1964). Circulatory and respiratory adaptation during long term non-steady state exercise in the sitting position. *Acta Physiologica Scandinavica*, **62**, 240-255.

Ekelund, L.-G., Holmgren, A., and Ovenfors, O. (1967). Heart volume during prolonged exercise in the supine and sitting position. *Acta Physiologica Scandinavica*, **70**, 88-98.

Granath, A., Johnson, B., and Strandell, T. (1970). Circulation in healthy old men, studies by right heart catheterization at rest and during exercise in supine and sitting position. In *Physical Activity and Aging*, pp. 48-79. Ed. by D. Brunner and E. Joki. Karger, Basel and New York.

Grimby, G., Nilsson, N. J., and Sanne, H. (1966). Repeated serial determination of cardiac output during 30 min. exercise. *Journal of Applied Physiology*, **21**, 1750-1756.

Guilford, J. P. (1965). *Fundamental Statistics in Psychology and Education*, p. 171. McGraw-Hill, New York.

Hartley, L. H., Grimby, G., Kilbom, A., Nilsson, N. J., Åstrand, I., Bjure, I., Ekblom, B., and Saltin, B. (1969). Physical training in sedentary middle aged and older men. III. Cardiac output and gas exchange at submaximal and maximal exercise. *Scandinavian Journal of Clinical and Laboratory Investigation*, **24**, 335-344.

Holmgren, A. (1956). Circulatory changes during muscular work in man. *Scandinavian Journal of Clinical and Laboratory Investigation*, Suppl. 24, 97 pp.

Jones, W. B., and Reeves, T. J. (1968). Total cardiac output response during four minutes of exercise. *American Heart Journal*, **76**, 209-216.

Kellermann, J. J. (1975). Rehabilitation of patients with coronary heart disease. *Progress in Cardiovascular Diseases*, **17**, 303-328.

Kennedy, C. C., Spiekerman, R. E., Lindsay, M. I., Mankin,

H. I., Frye, R. L., and McCallister, B. C. (1976). One year graduated exercise program for men with angina pectoris. *Mayo Clinic Proceedings*, **51,** 231-236.

Levy, A. M., Tabakin, B. S., and Hanson, J. S. (1961). Cardiac output in normal men during steady state exercise utilizing dye-dilution technique. *British Heart Journal*, **23,** 425-432.

Monroe, R. G. (1964). Myocardial oxygen consumption during ventricular contraction and relaxation. *Circulation Research*, **14,** 294-300.

Redwood, D. R., Rosing, D. R., and Epstein, S. E. (1972). Circulatory and symptomatic effects of physical training in patients with coronary heart disease and angina pectoris. *New England Journal of Medicine*, **286,** 959-965.

Saltin, B., and Stenberg, J. (1964). Circulatory response to prolonged severe exercise. *Journal of Applied Physiology*, **19,** 833-838.

Sarnoff, S. J., Braunwald, E., Welch, G. H., Jr., Case, R. B., Stainsby, W. N., and Macruz, R. (1958). Hemodynamic determinants of oxygen consumption of the heart with special reference to the tension-time index. *American Journal of Physiology*, **192,** 148-156.

Scheur, J., Penpargkul, S., and Bhan, A. K. (1974). Experimental observation on the effects of physical training upon intrinsic cardiac physiology and biochemistry. *American Journal of Cardiology*, **33,** 744-751.

Scheur, J., and Stezoski, S. W. (1972). Effect of physical training on the mechanical and metabolic response of the rat heart to hypoxia. *Circulation Research*, **30,** 418-429.

Sowton, E., and Burkart, F. (1969). Haemodynamic changes during continuous exercise. *British Heart Journal*, **29,** 770-774.

Sturzenhoffecker, P., Schnellbacher, K., and Roskann, H. (1976). Cardiac output and filling pressures at rest and during exercise. In *Proceedings of Int. Boehringer Mannhein Symposia—Ventricular Function at Rest and During Exercise*, pp. 26-28. Ed. by H. Roskann and Ch. Hann. Springer Verlag, Berlin, Heidelberg, and New York.

Wade, O. L., and Bishop, J. M. (1962). *Cardiac Output and Regional Blood Flow*. Blackwell Scientific Publications, Oxford.

Requests for reprints to Dr J. J. Kellermann, Cardiac Evaluation and Rehabilitation Institute, The Chaim Sheba Medical Center, Tel Hashomer, Israel.